Case Report

GIULLAIN BARRE SYNDROME ASSOCIATED WITH BRUCELLOSIS

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ABSTRACT

Guillain Barre Syndrome (GBS) is a prototypical post-infectious autoimmune disease. We report a case of Guillain- Barre syndrome in a 9-year old boy who was admitted with weakness of both lower limbs for 10 days and a history of previously treated brucellosis.

KEY WORDS: Guillain Barre Syndrome, Brucellosis, Autoimmune disease, Molecular mimicry.

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INTRODUCTION

Giullain- Barre Syndrome (GBS) is an acute inflammatory polyneuropathy which is most commonly characterized by rapidly progressive, essentially symmetric weakness and areflexia.[1] Since the marked decline in poliomyelitis incidence, GBS has become the most common cause of acute flaccid paralysis in children; development of GBS is in two-thirds of cases preceded by acute infection, typically with gastrointestinal or respiratory infections. Infectious agents related to GBS include cytomegalovirus, Epstein-Barr virus, Campylobacter jejuni, Mycoplasma pneumoniae and Haemophilus influenzae.[2] Although its pathogenesis is not clear, molecular mimicry seems to be responsible for GBS development after infection through the synthesis of autoantibodies against myelin gangliosides.[3] Here we report a 9-year-old boy with Giullain Barre syndrome and a history of recent documented brucellosis. GBS preceded by brucella organism has rarely been reported.[4-6]

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CASE REPORT

A previously healthy 9-year old boy was admitted on 23 June 2008 to our hospital because of progressive ascending weakness, beginning 10 days before admission. The patient was residing in a small village near Marand, a city in North West of Iran. On admission, he was alert and afebrile. He could not stand up, but sit without support; deep tendon reflexes were absent in all extremities. No sensory abnormalities, meningismus, and cranial involvement were noted.

He complained of paresthesia and pain on his legs. We first suspected acute GBS. Cerebrospinal fluid (CSF) examination revealed a raised protein level (172mg/dl), a normal glucose level (74mg/dl) and no cellularity. CSF culture did not yield any bacterial growth. Nerve conduction studies were suggestive of demyelinating polyneuropathy. The diagnosis of GBS was confirmed. Serum IgG and IgM

antibodies to campylobacter jejuni were negative, serum Anti-GM1 IgG antibody was positive. The patient did not give history of gastrointestinal or respiratory infections during last six weeks; however his mother gave a history of night sweating and non-specific malaise about six weeks before admission.

By diagnosis of brucellosis, he had received a course of antibiotic therapy. Therefore because of the occupational risk factors (he was grown in a rancher family) and a history of the ingestion of unpasteurized goat's milk and cheese, on suspicion of brucellosis serologic tests were done. Wright Agglutination tube test titer was 1/160, coomb's Wright titer was 1/320 and 2-mercapto-ethanol titer was negative, CSF Wright titer was negative and the results of routine laboratory studies of blood and urine were normal. This pattern of serology suggests that there is no active brucellosis, and antimicrobial treatment is not indicated. During hospital admission, plasmaphresis was done as the therapeutic approach in 4 sessions on every other day. On discharge he was able to walk with support .On follow-up visit (2 weeks after discharge) he was able to walk without aid, and at two months he could run normally.

DISCUSSION

This case documents the development of post-infectious autoimmune polyneuropathy which occurred after an infection that does not normally have this consequence. His ascending symmetric weakness, absence of DTRs and history and physical examination were compatible with GBS. Electrodiagnostic findings and albuminocytologic dissociation of CSF were supportive for the diagnosis.

The nervous system involvement in brucellosis can be categorized into central and peripheral forms. The former is usually acute and presents as meningoencephalitis, while the latter may either be acute or chronic in presentation. The peripheral nervous system involvement often presents itself as polyradiculopathy and less commonly as cauda equina like syndromes and peripheral mononeuritis. The infection may trigger an immune mechanism leading to demyelination (GBS).[7]

There are a few reports of GBS associated with brucellosis. One of these studies reported the case of a 14-year-old girl with Guillain-Barré syndrome associated with brucellosis due to Brucella melitensis[4] and a case of 9-year-old girl suffered from protracted paroxysms of severe hypertension before she developed classical signs of Guillain-Barré syndrome. Significant brucella antibody titres were found in the serum and complete recovery was observed after appropriate therapy.[5] Garcia et al reported three patients with GBS during active brucellosis, one consistent with axonal form and two others with demyelinating character of the disease.[6]

Molecular mimicry is an important mechanism by which infectious agents may trigger an immune response against auto-antigens. Gangliosides are composed of glycosphingolipids with one or more sialic acids linked to the carbohydrate moieties. Anti-GM1 IgG antibody is positive in about 30% of GBS occurring after C. jejuni infection. Anti-GM2 IgM antibody is positive in 10% of GBS occurring after cytomegalovirus infection.[8]

As GBS-associated C. jejuni expresses GM1 like lipooligosaccharides on its surface, it was thought that other GBS-associated bacteria might also express similar molecules on their surface. Watanabe et al found that GM1 ganglioside-like molecules were expressed on the surface of B. melitensis. They also noted that immunization with B. melitensis induced the production of anti-GM1 ganglioside antibodies and flaccid limb weakness in BALB/c mice. The development of GBS is thought to result of molecular mimicry between the outer core structures of bacterial ipooligosaccharides and human gangliosides.[9] As mentioned in our case description serum anti-GM1 IgG antibody was positive.

This case suggests that brucellosis should be considered as a preceding event for Guillain-Barré syndrome in the endemic areas for brucellosis and serological tests for brucellosis should be performed.

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